Correspondence

Serum PABA test in chronic pancreatitis

Sir.—We read with great interest the excellent article written by Dr Lang and colleagues about the value of serum PABA as a pancreatic function test (Gut 1984; 25: 508–12). The authors, in spite of the limited number of patients investigated, reported results suggesting a better diagnostic value in chronic pancreatitis of serum PABA analysis at 120 minutes after BT-PABA administration than the urinary PABA test. They also recommended a combination of the urinary and serum determination to increase the diagnostic value of this pancreatic function test.

To further support the validity of their conclusions we would draw your attention to our experience with urinary and serum PABA test in patients with chronic pancreatitis. We studied 14 adult subjects: seven healthy volunteers and seven patients with chronic pancreatitis (four with mild chronic pancreatitis and three with severe chronic pancreatitis); the degree of chronic pancreatitis was determined by clinical features, presence of steatorrhea, and secretin-cerulein test according to Gullo et al.1 Drug administration was discontinued three days before the beginning of the study. After a night fast, a basal sample of blood and urine was collected and 1 g of BT-PABA solution (Farmitalia, Milano), equivalent to 339 mg of pure PABA, was given with an appropriate stimulating meal. The urine was collected in a single specimen over eight hours. Urinary PABA was tested according to Yamato and Kinoshita.2 Blood samples were withdrawn after 30, 60, 90, 120, 180, and 240 minutes from the beginning of the test. Serum determination of PABA were carried out by the method of Bratton and Marshall, partially modified by us.3

The Table shows serum PABA values in the two groups and the statistical analysis of the differences. At 90 and 120 minutes the results of healthy subjects and patients with chronic pancreatitis are completely separated, whereas partially or completely overlap at the other time intervals. The trend of the serum PABA values is more evident in the Figure. In chronic pancreatitis a plateau is reached after 120 minutes with values about half of normal peak values which are reached after 90–120 minutes. In detail in the control group peak values were found after 90 minutes in four subjects, and after 120 minutes in the other three subjects, whereas all the chronic pancreatitis peak values were spread from 120 to 240 minutes.

Table Serum PABA values (μg/ml), reported as mean±SD, in healthy subjects (HS) and in patient with chronic pancreatitis (CP). Statistical analyses were performed with the Wilcoxon’s rank sum test.

<table>
<thead>
<tr>
<th>Minutes after PABA administration</th>
<th>30</th>
<th>60</th>
<th>90</th>
<th>120</th>
<th>180</th>
<th>240</th>
</tr>
</thead>
<tbody>
<tr>
<td>HS</td>
<td>2.6±1.2</td>
<td>5.0±2.1</td>
<td>6.9±2.0</td>
<td>6.8±1.7</td>
<td>5.9±2.1</td>
<td>3.1±0.5</td>
</tr>
<tr>
<td>CP</td>
<td>1.3±0.9</td>
<td>2.7±1.5</td>
<td>3.3±1.3</td>
<td>4.1±1.2</td>
<td>4.1±0.8</td>
<td>4.0±1.6</td>
</tr>
<tr>
<td>p</td>
<td>ns</td>
<td>ns</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>ns</td>
<td>ns</td>
</tr>
</tbody>
</table>

Figure PABA serum concentration at various specific times after administration of BT-PABA in controls (n=6, open circles) and in patients with chronic pancreatitis (n=6, closed circles).

Results of the urinary PABA test showed that four out of the seven patients with chronic pancreatitis had an abnormal urinary recovery (lower than 60%). In the other three patients with mild chronic pancreatitis the percentage of PABA recovery was greater than 60%, while in one healthy subject it was abnormally low.

Our results strongly support the data of Dr Lang and colleagues and confirm the validity of the serum PABA test in the diagnosis of chronic pancreatitis. We think, in partial disagreement with the authors, that the two hours blood sample alone, as suggested, is not sufficient. Two blood collections, at 90 and 120 minutes allow calculation of the serum peak value which gives the best discrimination between controls and subjects with chronic pancreatitis. We recommend serum PABA test with peak PABA determination not only in children, as we previously pointed out,3 but also in adults as a valid diagnostic
test for the evaluation of the exocrine pancreatic function.

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References

Coeliac disease presenting with intestinal pseudo-obstruction

SIR.—A coeliac patient presenting with intestinal pseudo-obstruction was recently reported by Dawson *et al* *(Gut* 1984; 25: 1003–8). Their case history is a good example of the possibility that coeliac sprue presents itself as intestinal stasis. Their allegation that pseudo-obstruction is exceptional in untreated coeliac sprue prompted us to the following comment.

In a group of 47 coeliac patients1 symptoms of ileus led in two patients (before the diagnosis coeliac sprue was made) to an exploratory laparotomy. No mechanical obstruction was found. Hypokalaemia was also ruled out. The intestinal mucosal biopsy was in both patients characteristic for coeliac sprue (no villi, hyperregenerative crypts). Both patients had a good reaction to gluten withdrawal. Symptoms of intestinal stasis never recurred. In one of the patients, who succumbed to myocardial disease shortly after, we did find an extensive accumulation of ceroid pigment in the muscular layer of the whole intestinal wall. In the other patient a marked deposition of ceroid was observed in the smooth muscle cells in a rectal biopsy specimen. The biopsy specimens from the small bowel by a Crosby capsule showed a musculiars mucosa without ceroid deposition. This means that the mucosal muscle cells are not always representative of the situation in the muscular layer of the intestine. Both patients had a very low vitamin E level (3.5 μmol/l and 4.0 μmol/l; normal range 25–35 μmol/l). We postulated that ceroid accumulation, resulting from vitamin E deficiency,2 may play a role in the aetiology of intestinal paralysis in coeliac sprue.

With regard to our own observations we are very interested to know if Dawson *et al* looked in their patient for ceroid accumulation or vitamin E deficiency.

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References

Books

Recent developments in alcoholism: vol 2. Edited by Marc Galanter. (Pp. 427; illustrated; $52.50.) New York: Plenum Press, 1984. This official publication of the American Medical Society on Alcoholism and the Research Society on Alcoholism is the second volume in the series and as one might expect from such an organisational background, takes good account of the ever increasing array of scientific disciplines involved in alcohol research. In the preface, Richard Deitrich pointed out that not only should such a volume serve the needs of the very broad interests of the research community but it should also provide a means for recruiting new investigators. In the reviewer’s opinion, these objectives are likely to be fulfilled for the topics covered are as fascinating as they are important. Section I is concerned with experimental social and learning models of drinking, where the emergence of addiction in relation to different patterns of drinking and various social factors are all critically considered and with all manner of fascinating data that the reviewer had not been exposed to before. Section II is concerned with alcohol and the liver: preclinical and clinical research. It is a first rate review by top workers of how alcohol injures the liver and the new work on oxygen metabolism is well covered. Section III relates to the important topic of aging and alcohol problems. This must be compulsive reading for all those concerned with health care provision for this rapidly expanding part of our population. The final section of contributions from anthropology to the study of alcoholism has much information of